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DIPHTHERIA—A CLINICAL STUDY.

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EMPHASIZING the importance of diphtheria and its perfect understanding by physicians, it is only necessary to consider for a moment its constant prevalence and its fatality, as shown by the mortality reports of all cities. In Denver, for example, it has, during the past five years, caused from five to ten per cent of the total number of deaths. The following table gives the exact figures:

Year.	Total deaths.	Diphtheria deaths.	Croup deaths.	Per cent. of deaths due to diphtheria.	Per cent. of deaths due to diph. and croup
1888	1729	77	43	4·5	7
1889	1808	77	28	4·3	5.8
1890	2530	210	50	8.3	10.6
1891	2118	145	30	6.8	8.25
1892[2]	1578	75	9	4·75	5·3

A rough estimate of the percentage of fatal cases may be made from a consideration of the number

[1] A paper read before the Denver and Arapahoe Medical Society, December 13, 1892.

[2] First eleven months.

of reported cases of sickness and the number of deaths. There were 440 cases of diphtheria reported during 1891, with 145 deaths, a mortality of 33 per cent.; adding croup there were 470 cases, with 175 deaths, a mortality of 38 per cent. During the first eleven months of 1892 there occurred 281 cases of diphtheria, with 75 deaths, a mortality of 26 per cent.; or, adding croup, there were 290 cases, with 84 deaths, a mortality of 29 per cent. The limits of this paper prevent any proper consideration of sanitary precautions for the prevention of diphtheria, which will be reserved for a future time.

The recent literature of diphtheria all tends toward a more certain understanding of its nature and more definite objects to be attained by treatment.

Jonathan Hutchinson voices the opinion of ultra-conservatism when, admitting the weight of the facts in reference to germs causing this disease, he says that he "is not yet convinced that they cause it." Bacteriologic investigators, on the other hand, are ready to prematurely credit the clinical phenomena to one germ only, and to call only those cases in which it is present "true diphtheria"—classifying all other cases, though clinically similar and equally fatal, as "pseudo-diphtheria," or as some other variety of anginose condition.

It is not unreasonable to assert that similar pathologic and clinical phenomena may be caused by a number of different organisms, existing separately or in company. No one of these germs is to be regarded as the sole and specific organism; one

may be less malevolent than the other; but the disease which it causes is clinically indistinguishable from that due to the other germ—it is equally contagious; the malevolent condition may at any time be added to the benevolent form; therefore, the disease is diphtheria, and is to be so classed for treatment and for hygienic precautions.

A fairly accurate definition is this: The disease-process or combination of processes known to us as diphtheria is both infectious and contagious. Its cause is an albuminous material of intensely poisonous character resulting from the growth, usually within the body or upon one of its surfaces, of any of the following microörganisms: 1, the bacillus diphtheriæ of Klebs and Loeffler; 2, the streptococcus diphtheriæ of Prudden; 3, a simple micrococcus resembling that described by Roux and Yersin.

These germs and the products of their activity give rise to a series of clinical phenomena so similar as to be practically indistinguishable. The ordinary genesis of the disease is probably as follows: 1. The germ obtains lodgment upon some mucous surface of the body, most usually in the upper respiratory passages, as a rule upon the posterior pharyngeal wall, in the palatine folds, on the tonsils, or in the nose (these localities are named in the order of frequency of primary involvement), and less frequently in the larynx. 2. The germ finding suitable pabulum grows and multiplies theron, and, as a result of its growth, the toxic albuminous body characteristic of the disease is formed and set free. 3. A series of local patho-

logic changes then occur, as follows: The epithelial covering of the mucous membrane is devitalized and cast off, and almost coincidentally a fibrinous exudate occurs, which, according to the rapidity of its formation, may or may not hold the dead epithelium in its meshes, and according to the concentration and virulence of the toxic albumin or "toxin," may or may not invade the basement membrane and deeper tissues underlying the mucous surface. 4. From this distinctly local disease-focus there now occurs absorption of all of the products of the pathologic processes there occurring; the toxic products of the diphtheria-germ (bacillus, streptococcus, or micrococcus, as the case may be) are accompanied into the blood and into the lymphatics by the toxins of those other swarming microörganisms which, as a rule, coexist, and which multiply readily in the necrotic tissues; sometimes the germs themselves find their way into the circulation, infecting both the blood and distant organs and structures.

As a result of this systemic invasion there occurs, usually, although not with absolute regularity, general malaise, anorexia, perhaps nausea and vomiting, rise of temperature, increased pulse-rate, lowered blood-pressure, and weakened heart-action; the excretory organs may be affected in a manner ranging from absolute stimulation to complete paralysis; the bowels are usually inactive. These clinical features are far from constant, and some of them are of little significance as compared with their relative importance in other diseases. Thus, in my experience, the rise of temperature means

almost nothing in diphtheria, while the circulatory changes are of supreme importance. Certain of the clinical changes are of greater or less intensity and of greater or less importance, according to the saturation of the system with pathologic products other than the specific toxins of diphtheria. It is certain that these other products, originating in a necrotic process are identical with those of ordinary septicemia; the clinical phenomena are exactly similar to those of ordinary septicemia, and emphasize the point which is of so great importance in reference to a proper understanding of the nature of diphtheria and its proper treatment, viz., that it is a septic disease. Or, perhaps a more accurate expression of the fact is that diphtheria pure and simple, a result of intoxication of the system with diphtheria-germs or their toxic albumins, is almost invariably accompanied by septicemia due to the absorption of other septic and necrotic products.

As already stated, the febrile conditions are not constant; neither are they found to exist coincidentally to such an extent as in other diseases; *e. g.*, the temperature may remain constantly normal or subnormal, while the pulse will range from 120 to 160 per minute. Then the rapid pulse of diphtheria is almost never full and bounding, but is almost always weak and compressible, often thready. Even when febrile conditions are marked there is a strange absence of delirium; in fact it is often noted that patients, especially children, suffering from fatal forms of diphtheria are rational up to the moment of death, thinking, speaking, and planning in the most connected and sensible manner, and, as a rule,

recognizing the approach of the great change and absolutely welcoming it. When the septic conditions are most intense, coma occurs. It has been determined that coma is probably due to an albuminous body existing in the germ and not formed as a result of its growth. This body is acid in reaction, of the nature of a protein, and differs radically from the toxins. The manner in which this body is set free—if it is set free—and the way in which it acts—if it does act—to produce the characteristic coma of diphtheria, are not as yet known. I am inclined to think that the further study of this problem and of the reciprocal actions and physiologic influences of these organic bodies may yet yield information of value in regard to the development of the antitoxin of diphtheria.

Returning, however, to the local lesion, which is worthy of extended observation and investigation, usually described and often existing as a yellow or grayish-yellow membrane, I have observed it more frequently in the beginning as a pearly-gray pellicle of extreme tenuity, often simulating the condition due to edema of the mucous and sub-mucous tissues. At other times a tiny patch resembles muco-pus filling a crypt in one of the tonsils, or hidden in the palatine recess, from which as a center it spreads quickly. Again, a slender, filmy, gray line appears first, crossing the tonsil at an irregular angle or hidden in one of the faucial folds. A favorite location is the posterior surface of the naso-pharynx, where the germ is really most likely to lodge, impinging there at the point from which the incoming air-current is deflected downward. The beginning membrane is not readily

discovered at this point, being hidden from view by the soft palate. Vocalization and the coincident rise of the palatal curtain will often reveal a hitherto hidden patch of membrane.

The rapidity of the extension of the membrane is quite variable. In some few cases the first-observed patch, film, or line, will remain stationary and of doubtful character for as long a period as thirty-six hours; more usually it spreads, so that in twenty-four hours an area of one or two square inches is covered; while more rarely it increases with alarming rapidity in the course of a few hours, so that a spot which in the early afternoon seemed insignificant or of doubtful character has by early evening become a dense necrotic mass, filling the whole of the fauces, obstructing the posterior nares, preventing deglutition and interfering with respiration. The little patient sits up in bed with the mouth open, partly for facility in respiration, partly because the swelling of the tonsils and cervical glands has become so great and so painful as to prevent the closing of the jaws. Thus in a few hours the local condition may become so serious as to endanger life, even although the systemic invasion has not yet been great. Cases of this class are, as a rule, rapidly fatal. The membrane thus early obtaining vantage-ground, extends rapidly both upward and downward, the nose becomes filled with a dense fibrinous mass, totally occluding its caliber and of such consistency that it can with difficulty be bored through with an ordinary probe; laryngeal stenosis occurs early, and the patient dies in less than seventy-two hours, regardless of the means adopted for relief. Intuba-

tion and tracheotomy seem to be equally valueless in these overwhelming cases, for after the first twelve hours the absorption of toxins and of septic material from the great necrotic mass covering and filling pharynx, nose, and larynx, is so great and so rapid that the blood is practically disorganized, the nervous system overwhelmed, the heart enfeebled beyond aid, and death is almost inevitable.

Rapid membrane-extension, such as is here described, is probably dependent upon three causes; (*a*) intensity or virulence of the poison, which may be due to high vitality or activity of the germ or to an especially favorable medium existing for its growth, the secretions of the individual being at the time in condition to furnish a good cultivation-fluid; (*b*) erosion of the epithelial covering of the mucous membrane; this condition may pre-exist or may be furnished at the time by ill-advised local medication with remedies which devitalize the epithelial cells and thus denude the mucous membrane; (*c*) idiosyncrasy of the patient. In some families, diphtheria occurs at periods remote from each other, the hygienic conditions differing at each time, but the type of the disease in the family is almost invariably the same, and the patients almost invariably die. The members of some families possibly have a lowered resisting power, both of the local cells to the germ, and of the system to the poison. The recurrence of the malignant type of the disease in some families can only be explained by some such supposition. I have observed that the members of such families, while otherwise perfectly

healthy, seem to have a tendency to catarrhal inflammations of the mucous membranes of the digestive and genito-urinary tracts as well as of the respiratory passages.

The usual factors of fatality-causation in diphtheria may be stated as follows:

1. Extensive involvement of mucous surfaces.
2. Rapid absorption of disease-products and consequent constitutional poisoning.
3. Laryngeal stenosis.
4. Pre-existing conditions that lessen the resisting power of the system.

Practically, the second and third factors are dependent upon the first, viz.: Extensive involvement of mucous surfaces; and the preponderating influence of this factor in causing death is very forcibly shown in the accompanying analysis of cases (see table on next page) in which I have preserved a record of the extent of membrane-formation.

The causes of death were as follows: Septic intoxication, 8; laryngeal stenosis, 7; cardiac paralysis, 6; hemorrhage from the bowels, 1; suppurative nephritis, 1; unknown, 2; total, 25.

In the one hundred and twenty-eight cases, operative interference on account of laryngeal stenosis seemed to be demanded, and was proposed, in nine cases. Five declined operation, and all died, three of the stenosis and two of septicemia. One accepted, but operation was deferred, and ultimate recovery ensued without it. Three were operated upon, two by tracheotomy and one by intubation, and all died. Each of these patients was practically moribund before operation was permitted.

CASES NOTED—128.

Location of membrane.	No. of cases.	No. of deaths.	Per ct. dying.	Per ct. of all.
Fauces only	44	1	2	34
" and palate	29	1	3.5	23
	73	2	3	57
Fauces and tongue	3	0	0	2
" and nose	24	4	16.67	18
" and larynx	2	2	100	1.67
" nose, and larynx	15	12	80	11
" " larynx, and ears	1	1	100	—
" " and eyes	3	2	66	—
" " larynx, eyes, and ears	1	100	2	—
" " and bowels	1	1	100	—
Palate only	2	0	0	—
Tongue only	1	0	0	—
Membrane not visible	2	0	0	—
	128	25	19.5	
Nose involved	45	21	47	35
Larynx involved	19	16	84	15
Eyes "	4	3	75	3
Ears "	2	2	100	—
Bowels involved	1	1	100	—
Cases not treated	11	7	64	—
" treated	117	18	15 33	—

It will be noted that only three laryngeal cases recovered, and that of the fatal cases, all but four had nasal and all but nine had laryngeal involvement. That this fatality is largely due to the extent of membrane from which absorption can take place is evidenced by the fact that only seven deaths occurred from a local cause (laryngeal stenosis), while sixteen deaths were due directly or indirectly to septic conditions.

In only five cases, or four per cent. of the number of which notes were preserved, were the fauces free

from membrane ; in two of the five, no membrane was visible at any time ; in two it appeared only on the hard palate ; and in one it was visible only under the tongue.

Of eighty-one cases in which membrane was limited to the fauces, the palate, and the tongue, but two died ; while of forty-seven cases in which the nose and larynx were involved, twenty-three, or forty-nine per cent. died. Of nineteen having laryngeal membrane, seven died of stenosis ; deducting these, we find that nine of twelve died of septic intoxication and cardiac paralysis, conditions due directly to the absorption of material from the local disease-focus. Of the nasal cases, twenty-one of forty-five (or forty-seven per cent.) died, a somewhat lower rate on account of the greater accessibility of the nasal cavity to treatment, but still a large number of deaths.

The four cases in which the membrane is recorded as appearing on the tongue are of special interest. The first was in a child two years old ; the disease was ushered in by a severe convulsion, during which he bit the tongue almost in two. The membrane developed on the wounded surface and on the dorsum of the tongue. The other three cases were all in adults and all in one family ; the membrane was a well-defined persistent patch under the tongue. Each of the patients had previously nursed a patient ill with the disease, and, although warned of the danger, persisted in reading books and magazines that had been in the sick-room from the beginning of the illness in the house. They had the habit of moistening the thumb under the tongue, prepara-

tory to turning the leaves of the book. All four patients recovered.

The four cases in which the eyes were involved were of especial interest, as the process seemed in each case to spread through the lachrymal duct. Three of them died when the palpebral conjunctiva had been invaded ; the fourth one recovered, affording me the undesirable opportunity of observing the progress of the destructive processes in both eyes. The left eye was first involved, membrane-formation having evidently taken place already in the duct, which was occluded from distention of the sac. The lower lid and then the upper one was invaded, ollowed by the ocular conjunctiva, which was rapidly covered, the membrane advancing with rapidity from periphery to center, suddenly choking off the nutritive supply of the cornea and causing it to slough forty-eight hours after the first invasion of the eye.

The sequence of events as observed on the conjunctiva of the lids was as follows: An intense hyperemia, then, twelve hours later, a roughened, granular appearance, with apparent epithelial desquamation ; this being quickly followed by marked anemia (perhaps only the deceptive appearance of the forming translucent veil of membrane). Twelve hours later there was a well-formed membrane, about one-sixteenth of an inch in thickness, lining the entire surface of both eyelids, and beginning to push on to the ocular conjunctiva.

In the right eye the changes were a little slower, but in practically the same order. Increased intra-ocular tension was noted by Dr. Chase, who had

kindly attended the case with me, and immediate preparations were made for paracentesis; but, before we could return, rupture had occurred, and the cornea also ultimately sloughed in part.

When recovery from the general disease occurred it was noted that a small portion of the lower segment of the cornea remained, and there was a dim perception of light. Dr. Chase kindly kept the eye under treatment, and proposed making an effort to form a new pupil, but the family removed to St. Louis, and all trace of the patient was lost.

This, and one of the fatal cases involving the eyes, had scarlatina immediately antecedent to their diphtheria, and the nose was seriously involved in all four cases.

The two fatal cases, in which membrane appeared in the ears, were also accompanied by nasal involvement, and the membrane extended through the Eustachian tubes, perforated the drum-membrane, and pushed out through the external meatus. Several other cases—all fatal—had middle-ear suppuration and perforation as a result, but a well-defined membrane was only detected in the two cases recorded.

The case of diphtheric enteritis recorded was under my personal observation, but not attended to by me. The patient was eleven years old, and the membrane was present in the fauces and in the nose. Intestinal hemorrhage was first noted on the fifth day of the disease, increasing in amount and frequency until death occurred on the morning of the seventh day. The blood was dark-colored and had the same foul odor noticed on the breath in those

cases in which the faucial membrane is thick and gangrenous.

One death, credited to laryngeal stenosis, occurred under the following circumstances: There was no membrane in the larynx, but an extensive and tough one in the fauces. About the sixth day this began to separate; a large piece loosened spontaneously, flapped down, was drawn into the larynx during inspiration, and the boy choked to death.

In two instances I have observed erysipelas coincident with diphtheria, and one of these patients, a young man, seventeen years old (Case LX), recovering perfectly from both his erysipelas and diphtheria, was immediately attacked by characteristic typhoid fever with all the classical signs and symptoms: rose-spots, typhoid tongue, sordes, typhoid temperature, tympanites, and ileo-colic pain, dying of intestinal hemorrhage in the third week of the typhoid fever.

Diphtheria is a disease of childhood rather than of adult life, as the following analysis of cases according to age will show:

1 year and under,	8 patients.	5 died.	62.5 per cent.
Over 1 year, not over 2,	12 "	3 "	25 "
" 2 years, " 3,	9 "	3 "	33.3 "
" 3 " " 4,	10 "	2 "	20 "
" 4 " " 5,	11 "	5 "	46 "
" 5 " " 10,	32 "	5 "	16 "
" 10 " " 15,	12 "	1 "	8 "
" 15 " " 20,	10 "		
" 20 " " 30,	16 "		
" 30 " " 40,	5 "		
45 years of age,	1 patient.	Recovered.	
47 " "	1 "	Died.	
52 " "	1 "	Recovered.	
	128	25	

Fifty of 128, or 40 per cent., were under five years of age, and 18 of the 50, or 36 per cent., died.

Eighty-two of 128, or 65 per cent., were under ten years of age, and 23 of them, or 28 per cent., died.

Ninety-four of 128, or 75 per cent., were under fifteen years old, and 24 of them, or 26 per cent., died.

There were 34 patients above fifteen years of age, and of them but one died. This was a man, forty-seven years of age, of dissipated habits, the disease well advanced before he came under treatment, and he died suddenly of cardiac paralysis when apparently convalescent.

The causes of increased prevalence and increased mortality among children are various. A sore-throat is usually not recognized and treated so early in a child as in an adult; treatment, when begun, is more difficult to carry out efficiently and thoroughly; the resisting power of the child's organism is less than that of the adult. The preponderance of fatality among very young children (under one year) is very marked, especially when we consider their comparative immunity from the disease. It will be noted that there were but eight cases of one year old and under. The youngest patient in the series was eight months old. It is a comparatively rare occurrence for nursing babes to contract diphtheria, even when exposed to it. But of those who do take it an extremely large percentage die; five of eight such cases, or 62.5 per cent., under my own care, died. Most of these very young patients live but a short time after their illness is recognized.

THE RELATIONSHIP OF SCARLET FEVER AND DIPHTHERIA.

The streptococcus diphtheriæ of Prudden cannot be distinguished by its physical characters from the streptococcus that causes scarlet fever, or from the one that causes erysipelas. It is not impossible that the same germ may have a different life-history under different circumstances; that environment may cause it, after many generations, to be endowed with new properties, malevolent or benevolent, as the case may be.

It seems probable that the streptococcus of diphtheria more commonly occurs in connection with scarlatina, and one observer (Heubner) is quoted as maintaining that what he calls "scarlet-fever diphtheria" is not so severe as ordinary diphtheria, that the tissue-necrosis is not so marked, and that laryngeal stenosis and paralysis very rarely result from it.

In the cases upon which this study is based there were 23 that occurred coincidently with or immediately following scarlatina, and 6 (25 per cent.) of these cases died, all but one evidently dying of the diphtheria. The nose was involved 9 times in the 23 cases and the larynx 4 times. In addition, 15 persons who had been exposed to scarlatina or to scarlatina diphtheria contracted diphtheria evidently of the common form, without any scarlatinal complication, and of the 15 one died. The nose was involved 3 times, and the larynx not at all in these 15 cases.

I have been told that in Bremen and Stockholm there occurs an overwhelmingly large number of the

variety of cases which require intubation or tracheotomy in order to avoid a fatal termination of the case within a few hours of the onset of the disease. The percentage of such cases occurring here is small. The literature of the subject seems to show that in New York and Boston operative interference on account of laryngeal stenosis is called for oftener and earlier than it is in Denver. It seems possible that these cases are of the variety due to the more malevolent germ, said to be the Klebs-Loeffler bacillus, while a great portion, though of course not all, of the cases of slower onset and milder manifestations, are due to the less malevolent germ, probably the streptococcus.

The grayish or yellowish-white pellicle that so often covers the tonsils from about the third to the seventh day of scarlet fever may not always be a diphtheric membrane; yet I am inclined to think that it usually is, and when the pellicle spreads on to the hard palate and clings there persistently for many days, recurring as quickly as it is removed; when it thickens into a grayish mass, pultaceous on the surface and harder beneath, and accompanied by its peculiar sickening odor of decomposition; when, preceded by an acrid discharge which erodes both mucous membrane and skin, it enters the nose and there thickens until it occludes the whole cavity, perhaps pushing—ashy or even black in color—out of the nostrils on to the lip; when it creeps through the lachrymal duct out of the puncta, lines the conjunctival sac, and chokes off the nutrient vessels of the cornea; when it passes along the Eustachian tube, breaks through the tym-

panic membrane and makes its appearance in the external ear; when it passes down into the larynx, covers the cords, and, occluding the glottis or slipping down in thinner form, invades the bronchi to their minutest subdivisions; then, whether it has preceded, or succeeded, or accompanied scarlatina, I recognize it as diphtheria, capable of spreading the contagion of diphtheria, with or without any accompanying disease, and of communicating diphtheria to persons who have previously suffered from and thus been made immune to the accompanying scarlatina.

There is a variety of deposit on the tonsils during scarlatina which, developing in the throat at the height of the anginose inflammation, disappears in a few days, never having at any time attained any great thickness, not spreading to any great extent, and usually capable of being easily removed with a swab. This I have not classed as diphtheria. But membrane of the more truly diphtheric type often occurs at this time, or its occurrence may antedate the appearance of the rash by several days, or even a week; or again, membrane may suddenly make its appearance during the eruption, or after the efflorescence has subsided and desquamation has far advanced.

It is peculiar that this truly diphtheric process occurs often in persons who have only been exposed to ordinary scarlatina, there being no diphtheria existing at the time in the neighborhood, the patients affected having previously suffered from scarlatina. Examples of this fact are Cases VII, XXVI and XVII of the series reported in this paper.

They were exposed to scarlatina simplex, but took diphtheria; one developed a rash four days after having membrane in his throat. Cases IV and V, who had previously suffered from scarlet fever, contracted simple diphtheria of a malignant type from such an exposure. Case LXXV, a mother who had suffered from scarlet fever in childhood, nursed her children (Cases LXXIII and LXXIV), suffering from scarlet fever complicated with diphtheria, and contracted diphtheria alone. Both children died of cardiac paralysis. The mother had membrane in the nose and on the palate to within an inch of the teeth, had palatal paralysis as a sequence, and was a long time in regaining her strength.

It would have been interesting to have had a bacteriologic examination in these three cases to determine whether the infection was from the streptococcus, or the bacillus, or from both at once.

Case CV was a woman who had scarlet fever in childhood, nursed her two children through scarlatina simplex, and was immediately taken down with a severe type of diphtheria.

Cases CVI and CVII were a mother and child in a family four other members of which had just recovered from simple uncomplicated scarlatina. The fifth child, exposed only to the scarlatina, contracted both this disease and diphtheria. The mother contracted diphtheria alone.

Case CIX contracted diphtheria from Case VIII, which was one of laryngeal diphtheria.

Case CXV contracted diphtheria from Case CX, one of simple diphtheria of the fauces and palate.

These four cases were placed in the same room in the hospital; the room had not had a case of scarlet fever in it for many months, and it had been thoroughly fumigated with sulphur and scrubbed with corrosive sublimate solution about once each month for a year. And yet Cases CIX and CXV, exposed to and suffering from diphtheria, were simultaneously attacked by scarlet fever. Both had albumin in the urine. Both desquamated. Case CIX died of suppurative nephritis, having apparently entirely convalesced from both his diphtheria and scarlet fever. I have included this death in the series of cases herewith reported as due to diphtheria.

These clinical testimonies to the apparent relationship or affinity of the two disease-processes might be multiplied, but multiplication of this simple testimony does not elucidate anything. May we not hope that careful and systematic bacteriologic study of cases may yet enable us to understand the rationale of the relationship, and simplify diagnosis, treatment, and prevention?

THE IDENTITY OF MEMBRANOUS CROUP AND LARYNGEAL DIPHTHERIA.

The lessening frequency with which membraneous croup appears in our mortality reports seems to indicate that this disease is either dying out or that practitioners are calling the condition formerly so designated by another name.

In all of my professional work, as student, as hospital resident and as practitioner, it has never been my fortune to see, either in my own practice or in that of my professional friends, a case that I

could call one of membranous croup, *i. e.*, a non-infecting stenosis of the larynx, due to membrane-formation, as it is described in the text-books. I have seen cases that were called membranous croup, but with the diagnosis of which I did not and could not agree. The following cases are cited as examples:

Case I.—A young man now twenty years old. Every slight cold affects his throat. He has frequent attacks of tonsillitis and occasionally laryngitis of a catarrhal nature. His parents state that he had frequent attacks of greater severity in his earlier years, accompanied by a "croupy cough." He would sit down to a meal feeling perfectly well; an attack would come on while he was eating; and he would almost die. Dr. W—— said it was membranous croup, and often he arrived just in time to save Harry's life. This is a case in which the patient's present condition offers a lucid explanation of the frequently recurring attacks of "membranous croup" in his earlier years.

He was finally cured of his "membranous croup" by the frequent administration of a home-remedy containing alum, sulphur, and molasses.

Case II.—A child three years old, seen in consultation with Dr. Bryant. It had a slight cold, and was suddenly taken with difficult, "crowing" respiration, rough, croupy cough, the ribs drawn in and the interspaces not filling out.

In this case the symptoms seemed typical of what is ordinarily called membranous croup, and tracheotomy seemed inevitable. Small fine râles were discovered all over both lungs, and inhalations of water vapor and of slaking lime seemed to relieve the difficulty of breathing, so that operative procedures were postponed for a day.

At the end of this time the laryngeal symptoms had greatly ameliorated, and disappeared entirely after a few days. No membrane was ever expectorated, but there was considerable bronchitis, with mucous expectoration. The case was plainly one of catarrhal laryngitis, and our diagnosis of membrane obstructing the larynx was premature and a mistake.

Case III occurred in a child just removed from a day-nursery, where it had been in the same room with four other children, all of whom had been attacked with diphtheria, and all of whom died. Two of the four had membrane in the larynx as well as in the fauces, and the other two had nasal involvement. This case, the fifth child, was suddenly seized with laryngeal symptoms and died after a very short illness. The attending physician certified the cause of death to be membranous croup. It seems to me almost certain, when I consider the intimate association of the patient with the four fatal cases, and its death in a few days after theirs, that the real case of death was laryngeal diphtheria.

Case IV.—The first of the four fatal cases referred to as being removed from the day-nursery. The attending physician informed the board of managers of the institution that it was suffering from membranous croup, and they, in consequence, were greatly amused at the precautions taken by the Health Department to prevent the spread of the disease. The patient came under my care at quarantine, and was a typical case of diphtheria. The membrane involved the fauces, palate, and larynx, and the child died after seventy-two hours' illness, having been tracheotomized six hours before death, which was due to heart-paralysis.

Case V.—A boy two years old was suddenly attacked one evening with difficult respiration and croupy cough, having complained for a day of

what the parents regarded as a common cold. was called hurriedly, and found the child black in the face and almost dead.

I was tempted to tracheotomize at once, but the subcutaneous injection of apomorphine caused prompt emesis, and the child's respiration improved. Turpeth mineral and calomel were administered in one-grain doses every fifteen minutes; the vomiting continued, the spasm of the larynx relaxed, and he became much better. The throat was carefully examined, and no membrane could be detected; but in spite of this the child seemed critically ill. The next day examination revealed a typical diphtheric membrane that had appeared since the preceding evening and covered both tonsils. It spread rapidly, covered the palate and advanced into the nose. For five days the little patient's condition was extremely precarious; then improvement began, and the membrane entirely disappeared in thirteen days after the date of the original attack. After the first evening there was no apparent laryngeal involvement.

There can be no doubt that this case presented all the classical symptoms of membranous croup the evening it was first attacked, and if death had occurred (and it seemed imminent), most practitioners would have certified to that as the cause of death. The subsequent history of the case, however, showed that the initial attack of laryngitis was probably only functional, occurring as the ushering-in symptom of an attack of diphtheria.

Case VI.—A child, aged one and a half years, a brother of Case LI, which died two weeks previously. Another sister had also died of a most pernicious form of diphtheria one week before. This babe was nursed by its mother, who was also nurse for the two children who died. It was taken ill, and died in twenty-four hours of so-called "membranous croup."

Case VII.—Into a house next to one in which diphtheria occurred one year ago, the premises of both being in a very filthy condition, a new family moves. After a week one child is taken sick and dies in twenty-four hours with what the physician calls "pseudo-membranous laryngitis." The same day a second child is taken ill with the same complaint, and dies in thirty-six hours. In the second case there is membrane visible in the fauces. The physician calls it "contagious membranous laryngitis" but says it is not diphtheria, although he regards it as being "quite as contagious as diphtheria."

Cases similar to the last two might be multiplied almost indefinitely from the records of the Health Department; a death from "membranous croup" being reported immediately before or immediately after several cases of diphtheria have occured in the same house or in an intimately associated family. Perhaps these occurrences only emphasize the cautionary admonition of Cohen and Eshner in their recent work on medical diagnosis, that "until a physician acquires considerable experience he had best consider all cases of membranous croup as diphtheritic." Yet, in my experience, older practitioners more frequently than younger ones pronounce laryngeal diphtheria to be "croup," and I am inclined to regard the admonition quoted as catering too much to the fallacious belief which dignifies a symptom with the individuality of a disease.

One may not absolutely deny the possible existence of a non-infectious membranous laryngitis, for the condition would be a parallel one to that which exists in membranous bronchitis and enteritis. These

diseases are, however, wonderfully rare, while the majority of believers in membranous croup rate it as a comparatively common disease.

Very recently Fraenkel published a paper detailing the results of autopsies on four cases of so-called primary or non-infectious membranous croup. In all, the mucous membrane of the pharynx and tonsils was intact, the disease-process being limited to the larynx, and in all the laryngeal membrane was found to contain the Klebs-Loeffler bacillus, the identity of which was proved, first, by staining, second, by culture-experiments, and, third, by the inoculation of animals. Still later, Martin of Paris found the Loeffler bacillus in 21 of 34 cases of croup, in which there was no membrane in the fauces. *Query:* Were the other 13 non-diphtheric?

Very important, as bearing on this particular point, is Prudden's case: One child died of a disease pronounced by the attending physician to be "croup, and not diphtheria," and Prudden himself failed to find either the bacillus or streptococcus of diphtheria in the membranes. However, a few days later, a second child of the same family died, presenting all the constitutional and local signs of diphtheria, "and the streptococci were found, not only in the membrane but in the viscera." Even the failure to find the diphtheria-germ in all cases is thus shown to be not positive or final testimony as to the non-contagious character of the disease; clinical evidence is equally important, and is very properly a supplement to, or supplemented by, bacteriologic investigation.

THE RELATIONSHIP OF DIPHTHERIA AND FOLLICULAR TONSILLITIS.

Occasionally that common and readily-identified disease, follicular tonsillitis, occurs epidemically and in a somewhat modified form. It has been asserted that when it so occurs it is really diphtheria. It would seem that there could be but one method of settling this question, namely, by bacteriologic examination of a large number of such cases. The type of follicular tonsillitis is so common and so readily studied, that it is a matter of some surprise that such an investigation has not already been made.

Clinical arguments against the supposed identity of the two conditions are, however, almost conclusive. In families or in institutions in which follicular tonsillitis prevails, all the cases are follicular; none develops extensive membrane; the nose is never involved; none of the patients dies. It is possible for a patient with this disease to be seriously sick, but the febrile conditions are almost constant in their occurrence and relationship; high temperature for a day or two is common, and it accompanies a full, bounding, rapid pulse; delirium may be coincident; when the febrile conditions subside they do so by crisis; circulation and temperature return to the normal at once and together. What a contrast to the progress and relationship of the febrile conditions, as already described, existing in diphtheria?

Of course, it is possible for diphtheric infection to be added to a previously existing follicular tonsillitis, but in such a case the clinical picture will change quickly, and it will always be noted that any immediately succeeding cases are typically diph-

theric, both in incipiency and in progress. The follicular feature does not persist and manifest itself in subsequent cases after real diphtheria has made its appearance.

The deposit of follicular tonsillitis is a secretion, modified and unhealthy, perhaps, but still a secretion, and hence readily removed without harm to the underlying tissues. In sharp contrast is the diphtheric membrane, which is a fibrinous exudate upon a previously eroded surface, from which it cannot be removed without violence.

This distinguishing feature is all-important in the diagnosis, which is rarely difficult. If real doubt exist, twelve or twenty-four hours at most will suffice to clear it up. However, the fictitious credit attaching itself to the supposed rapid "cure" of cases of this kind has proved a strong incentive to some practitioners to pronounce as diphtheria all cases of follicular tonsillitis. This practice is most common in communities in which cases are not reported to the health-officers, and in which the premises are not placarded, and no quarantine is enforced.

A last argument against their identity is this: The diphtheric membrane ordinarily begins at one spot or location, from which, as a center, it spreads with a rapidity that is often alarming. Given a dozen or more foci (as would be the case if each follicle in the tonsil were a center from which the membrane could spread), and the rapidity of extension would be astounding. This would, indeed, be the most terrible and fatal form of diphtheria, by reason of the favorable circumstances attending its incipiency.

But the facts show that follicular tonsillitis is so seldom fatal that we may really say that it never kills, unless there exists some peculiar cachexia or other modifying condition of the patient.

The question of the identity of diphtheria and follicular tonsillitis may then be dismissed as unworthy of serious consideration.

TREATMENT.—The rational and successful treatment of a disease so varied in its manifestations and so various in its intensity as is diphtheria must not be entirely empirical, but must be based upon a knowledge of its pathology. The aim of treatment must be to combat those conditions or to abort those processes that result in death.

The modes of death from diphtheria may be classified with more or less exactness as follows:

A. *From systemic causes:*

1. Inanition
   - Non-assimilation
     - Inability to digest.
     - Inability to absorb.
   - Inability to partake
     - Paralysis of the muscles of deglutition.
     - Inflammation of the fauces.
     - Obstruction by membrane.
   - Neglect of feeding.

2. Septic conditions
   - High temperature. Rare.
   - Hemorrhage due to lack of vitality of capillary walls.
   - Coma.
   - Degeneration of the blood.

3. General paralysis. Very rare.

B. *From local causes:*

1. Stenosis of larynx,
   - By membrane.
   - By foreign body, on account of paralysis of muscles of deglutition.

2. "Heart-failure"
   - Fatty degeneration of heart.
   - Degeneration or inflammation of cardiac ganglia.
   - Disintegration of blood.
   - Endocarditis.
   - Paralysis of heart, due to central lesions of nervous system.

3. Pneumonia { Ordinary. / Septic. / Membrane-extension.

The conditions here outlined are, in their various modified or aberrant manifestations, almost legion; they may exist separately or at once, and a somewhat elaborate scheme of treatment is necessary in order to combat them. No hard-and-fast line can be drawn as to medicaments or dosage, for they may need to be varied almost from hour to hour. Few diseases—indeed, I am almost prompted to say, no other disease—demands so much or so varied medication, and in no other kind of sickness have I seen medication do so much absolute and incontrovertible good.

There is, however, no *specific* medication for diphtheria. No one remedy can cure all cases or keep up an uninterupted successful record during several successive epidemics or years. Nevertheless, it can be said without fear of contradiction that the general principles of therapeutics, faithfully followed, even in desperate cases, are here, as almost nowhere else, productive of results that are gratifying to both physician and patient.

Inanition must be combated by systematic attention to the diet. Easily-digested and easily-absorbed materials are a necessity, and should be administered with regularity at short intervals. Peptonized milk is of great value; beef-juice, freshly expressed, may be given frequently in small doses; the white of an egg, well beaten, with pepsin added, is easily retained, and may be given either by mouth or by rectum; oatmeal or cracked wheat will often be very grateful to the patient, and may be given freely.

The treatment of the septic conditions must aim (1) to encourage excretion, and especially by such methods as will carry off the toxic products that have been thrown into or formed in the blood ; (2) to increase the resisting power of the tissues and especially of the hematic elements ; (3) to antagonize the vitality and development of microörganisms, and (4) to destroy or neutralize the chemical products of germ-life.

The remedies that have given me most satisfactory results in meeting the indications just named are calomel, tincture of iron chlorid, whiskey, and mercury bichlorid or biniodid.

The effect of calomel on the system made it years ago the one great remedy in medical practice. An unintelligent prejudice has, during the past generation, kept it from being employed frequently or efficiently ; but we are again coming to appreciate the fact that "modification of the secretions and excretions" is an essential of successful medical treatment. Especially is this true of diphtheria, in the therapy of which disease calomel now holds a leading place.

Given in a dosage which seldom needs any limitation until its characteristic effect is produced, I have never seen calomel do harm. But, as the dosage is for effect, it should be discontinued or decreased when that effect, as indicated by thin, green bowel-movements, has been produced. Sometimes a total of ten grains, given in doses of one grain every hour, will be sufficient to produce the result. At other times as much as 200 grains will be required.

In answering the question: "What does calomel do and how does it do it?" one must depend partly on facts and partly on theory. The general condition of the patient is improved, the color becomes better, the pulse steadier and firmer, the tendency to coma is less, the bowels and kidneys both act more freely. In the throat it is observed that the spread of the membrane is limited or, at least, the rapidity of its spread is decreased; thick membranes become thinner and thin ones fail to become thick; extension of the disease into adjacent organs, especially into the larynx seems to occur but rarely after the system has been mercurialized. That this accounts for the lessening necessity for operative interference, on account of laryngeal stenosis, in the practice of those who depend upon mercurial treatment of the disease, I am thoroughly convinced.

The manner in which calomel, as well as other mercurials, such as biniodid, bichlorid, or cyanid, produces these effects, I take to be this: *a*, by a cholagogue action, the blood is relieved of toxic materials and the abdominal circulation is stimulated, thus preventing blood-stasis in the spleen and liver, where the final biologic product of the diptheria-germ is largely formed, such formation being favored by stagnation of the current; *b*, the sympathetic nervous system is relieved of the depressing influence of generally-diffused venous and capillary pressure due to stagnation of blood-flow; *c*, the heart itself is no longer gorged with unhealthy venous blood, and acts more freely and with greater force; *d*, the toxic materials in the blood and lymph have their activity lessened or

are partly rendered inert by the coagulating action of the mercurials; *e*, the germs themselves are destroyed and the toxic materials contained in them partially neutralized by coagulation; *f*, the changes thus brought about in the blood lessen the tendency to fibrin-exudation, and consequent membrane-formation at the local disease-focus.

It will be noted that this explanation covers not only the therapeutic action of the calomel, but the systemic effect of the mercurial salts. I am in the habit of giving the bichlorid or biniodid (there seems to be no choice between them) in solution or in tablet triturate form, in doses ranging from one-thirtieth to one-tenth of a grain, every one, two, or three hours. For a child of from two to five years old I begin with one-thirtieth of a grain every two hours, lessening the interval to one hour after a few doses, and doubling or trebling the dose at the end of the first or second day. The aim is to bring the system under the influence of the mercury as quickly as possible. When this is done the disease ceases to advance. Then I hold the medication at the dosage reached, or lessen the dose or increase the interval of administration as the case demands.

Of all the cases I have so treated only one was ptyalized. The patient was a female, aged twenty-six, to whom I had been giving one-sixteenth of a grain of mercury cyanid every hour for about one week. Membrane covered the fauces and the palate to within one-half inch of the teeth. In twelve hours the membrane disappeared synchronously with the unheralded appearance of severe ptyalism. There was no re-formation of membrane,

but the woman's teeth were loose for many months, and for weeks she had to take a simple "slop-diet." There was no necrosis, and the recovery was ultimately perfect.

Alcoholic stimulants are also of primary importance, here even in greater degree than in other septic diseases. No other remedy is known that is as potent and diffusible. If any other of equal efficacy is ever discovered alcohol may be given up, but not until then.

In the therapy of diphtheria this remedy serves a triple purpose: *a*, It is a stimulant of unequalled efficacy; *b*, it is a readily oxidized food; *c*, it is an antiseptic agent, both locally and systemically. In severe forms of diphtheria it is almost impossible to give too much of it. The dosage must be large, frequent, persistent; never at greater intervals than two hours, often at as short intervals as fifteen minutes, and sometimes, when the patient's vitality is at a very low ebb, its administration may be practically constant, the alcoholic stimulant being, properly diluted, continually administered by means of a medicine dropper.

It is frequently observed that the failing circulation regains its vigor, the sinking patient rallies, as the diffusible stimulant begins to be absorbed; while, as the effect of the dose wears off, the circulation again fails and vitality again becomes enfeebled. The amount of stimulant necessary is of course dependent upon the individual case, but must always be in excess of that given in other diseases. Many patients under six years of age require the administration of an ounce of whiskey every hour

for from two to six days, and to several I have given double that quantity, the patients recovering.

I have never yet observed the slightest harmful effect from the administration of alcoholics in diphtheria. In addition to its long-known stimulant properties, and its ready oxidation in the circulation, recent biochemic research has led to the conclusion that in diphtheria, at any rate, it has a distinct antiseptic action. The albumoses formed in the diphtheric membrane are precipitated and their absorption is prevented by alcohol. The germs themselves are devitalized and the toxic substances already absorbed are probably rendered partly inert by the action of the alcohol, after its absorption into the circulation.

Iron, in the form of the tincture of the chlorid, has long been a favorite remedy in all diseases accompanied by degeneration of the hematic elements. In diphtheria its beneficent effects have also long been recognized. But it is ordinarily given in too small doses. We must not lose sight of the fact that we are not dealing with a simple anemic condition, but with one of profound toxemia, which is acute in onset, malignant in action, and overpowering in its effects.

Remembering these facts, and the additional one that probably not more than half of what is given is absorbed, let us give iron in large enough doses to do good. To children from two to five years old I give from five to ten drops at least every two hours, often every hour; to adults never less than ten minims each hour, and frequently twice or thrice that amount.

The treatment of weak heart is but little different from that demanded for the same condition accompanying other diseases. Digitalis, nux vomica, and strophanthus are the indicated remedies, and to them may be added convallaria, nitroglycerin, caffeine, camphor, and musk; alcoholics are already being administered.

In those cases in which weak heart is practically due to hemorrhage it would seem that transfusion might be resorted to. At least the one-fourth per cent. saline solution should be injected subcutaneously or into the bowels. Such a case was No. CIX of those reported. The boy was eight years old and had apparently completely recovered from a severe attack of diphtheria in which the membrane affected the fauces, palate, and both cavities of the nose. He began to sink suddenly; vomited continuously; his pulse became thready, and disappeared at the wrist fourteen hours before death; his lips were ashen, and he complained of an intolerable thirst, but could not retain water in the stomach. Digitalis and nux vomica were freely given. Hot saline enemas were given, but failed to relieve, and he died thirty hours after the first sign of collapse.

The autopsy revealed only congestion and slight suppuration in each kidney, and about two pints of serous fluid free in the peritoneal cavity. These conditions did not seem to be sufficient to account for death. I have felt that the patient might have recovered if the saline solution had been injected into his veins. Certainly in another case like it I shall resort to the measure.

Albumin in the urine does not seem to me to be

an indication that is of any value as bearing upon treatment. In almost all cases the patient is already taking so much medicine for conditions of greater urgency that I do not see that any added medication will be justifiable. Nor does it seem, except in cases of suppuration like the one just cited, that albumin in the urine will have any bearing upon the final result of the sickness. During convalescence, should the albumin persist, I see no objection to the administration of some mild diuretic, like liquor ammonii acetatis. It seems probable, however, that the medication already described is good treatment for albuminuria, and as that is usually being given anyway, the detection or non-detection of albumin is a matter of indifference, so far as modification of treatment is concerned.

An additional remedy for systematic use is hydrogen dioxid. Patients whose lips are bluish-black, and with evidently impeded circulation, are benefited by its frequent free administration.

The ten-volume or fifteen-volume solutions of this drug, as found in the market, are innocuous even in excessive dosage, provided that they are pure. When there is an excesss of acid present, the drug will irritate or even denude the healthy mucous membrane. Of a pure specimen of the drug I give from one to four drams every hour, with the result in many cases of bringing a healthier pink color to lips and cheeks. The oxygen held in solution in the preparation is probably given up in the system, with the effect of promoting perfect oxidation and toning up the vitality of the red blood-corpuscles. Hydro-

gen dioxid should not be administered in metallic spoons, as they lessen or destroy its activity.

Local Medication.—Recognizing germs as the primary cause of diphtheria, local treatment is directed as follows:

1. Destroy the germs.
2. Neutralize their active products.
3. Limit membrane-formation and destroy what is already formed.

As has been stated, the remedies already recommended for internal administration have a local antiseptic and deterrent action, which must be exerted to some slight extent upon the local disease-focus as they pass over it. This slight action is, however, far from being efficient or sufficient. Energetic measures should be directed to the local trouble. Mechanical removal of the membrane has not given good results, as the violence necessary to accomplish it does harm to the underlying tissues, and the measure itself is ineffectual, new membrane appearing after a few hours. Destruction of the membrane and adjacent tissues by means of the actual cautery has been recently proposed. The proposition seems reasonable, but it is not feasible. The proportion of patients whose families would permit such an operation at a time when it would prove effectual is very small indeed. Cauterants, such as silver nitrate, ferric alum, carbolic acid, nitric acid, chromic acid and similar compounds, both in strong and weak preparations, have been urgently advocated by some practitioners as invariably successful in removing and preventing reformation of the membrane. I have used silver

nitrate and carbolic acid with but little success, and ferric alum with absolutely no success. The other agents proposed for this purpose seem so unsuitable that I have not used them.

Mixtures of iodin and carbolic acid of about 2 or 3 per cent. have been used with success by some practitioners, and I have ventured to rely upon them in a few mild cases, which fortunately recovered. The medication seemed to have no real effect upon the membrane.

The medicaments which have given me the most satisfactory results have been hydrogen dioxid (from three-volume to fifteen-volume solutions), corrosive sublimate, 1:2000 solution, carbolic acid, from 1 to 2 per cent. solution, and lime water. Trypsin and extract of pancreas I have used both in solution and in powder. While efficient, they are not to be compared to corrosive sublimate or to hydrogen dioxid sprayed with an atomizer. These two remedies I find of the greatest efficiency, and they may be used together without trouble. Except occasionally in adults, I have altogether stopped using the swab, as the violence which it does to the tender surfaces of the throat, and the struggles necessary in order to use it, are of great harm to young children, doing more damage than the local medication does good. The atomizer is, perhaps, the most efficient instrument for local medication. All of its tubes should be of rubber; the mouthpiece should be long and stout, the tip broad and with rounded end, so as not to injure the throat. The water-oil atomizer I find most satisfactory.

Very recently, Dr. Williams (Boston *Medical and*

Surgical Journal) has recommended the use of very strong solutions of hydrogen dioxid, from fifty to two-hundred volumes, for local application to the membrane. The argument in favor of this method seems most reasonable, but I have not yet had an opportunity to put it into practice. One posssible danger would seem to be that an agent of such a high degree of saturation would be a cauterant to the healthy mucous membrane.

Many authorities recommend the use of the atomizer, swab, or gargle, only every three or four hours. This seems to me, perhaps, as bad a recommendation as can be given. In severe cases no medication can be of effect in thinning the membrane or n preventing its spread, which is not repeated, at least every hour, and in a large number of cases it is necessary to spray the throat at least every half hour or every fifteen minutes. This will require the constant attention of the nurse, but it will often save the patient.

When the nose is affected it should be syringed frequently. For this purpose the same solution may be used as for the throat, but I have found that in the nose the 1 or 2 per cent. carbolic-acid solution is better tolerated by the patient. The wash should be used at least every hour. In forty-five patients with nasal diphtheria this treatment was used, and twenty-four of them (53 per cent.) recovered, while of those who died (twenty-one), two-thirds had laryngeal membrane as a complication.

Thin flakes of membrane, which persist for a long time after the general disease has abated, are best treated by some mild local caustic. The double

chlorid of gold and sodium, in 2 per cent. solution, has been of service to me in such cases.

Enlarged cervical lymphatics and enlarged tonsils, which occurred in about 10 per cent. of the observed cases, have yielded most readily and without suppuration, except in two instances, to inunctions of unguentum hydrargyri.

The sequelæ of diphtheria are quite varied. Transient peripheral paralyses are the most common, and may be named in order of frequency as follows: The palatal muscles; the ocular muscles; the peronei, and the tongue. Multiple palsies are rare.

One patient of mine, Case LXVII here reported, a child one year old, made a perfect recovery from the diphtheria, but died three months afterward with what was shown by the post-mortem examination to be a general myelitis. Dr. Eskridge observed this case and now has the specimen.

Since adopting the general methods of treatment which I have described, and especially since beginning the free use of mercurials, paralyses have been quite infrequent in my experience, and it seems probable that the treatment is to be credited with this result. One peculiar case of paralysis seems worthy of record here, as it at first seemed to be truly diphtheric, and illustrates, perhaps, a common error in diagnosis. An infant under observation by Dr. Sewall and myself had a severe attack of diphtheria, and immediately after recovery one leg was useless. Dr. Sewall diagnosticated diphtheric paralysis. I examined the babe, and, from a certain tenderness and fixation of the hip joint, diagnosticated accompanying incipient hip-

joint disease. The hip symptoms soon disappeared, however, and I "revised" my diagnosis and agreed absolutely with Dr. Sewall. Treatment seemed unavailing, and after several months we placed the child under Dr. Eskridge's care. His diagnosis was peripheral cerebral hemorrhage. Under appropriate treatment the cerebral clot was absorbed and the paralysis disappeared.

Diphtheric pneumonia sometimes occurs, and is usually fatal. The only cases that I have observed were two, both in children two years of age, and both of doubtful diagnosis. They were the first of the family to be taken sick. In neither case could I discover membrane, but both died within a short time. In each family there subsequently occurred several cases of undoubted diphtheria. My experience has thus not been in accord with the generally and undoubtedly accurately recorded cases in which the pneumonia has accompanied or followed the diphtheria.

The total number of cases of which I have preserved notes as to treatment is 117; all but about half a dozen of the mildest ones having been treated systemically upon the plan outlined in this paper.

Of the 117, but eighteen patients, or 15.3 per cent., died.

When it is taken into consideration that many of the cases were seen only after several days of neglect or of careless treatment, and that a comparatively large percentage of the cases were of the kind usually described as malignant, the death-rate is to be considered low. That the medicaments employed had something to do with the low death-rate I am

convinced; and the frequency of their administration is an item not to be overlooked. Indeed, if there is one point to be insisted upon with unyielding urgency, it is this of large dosage at short intervals in cases of diphtheria. The directions given by some authors to spray the throat every three or four hours, or to give iron or whiskey three times a day, or perhaps every four or five hours, are admirable examples of "how not to do it." It is to be hoped that that kind of teaching and of practice will soon pass away.

In conclusion, I wish to say that there are many other diphtheria remedies which I have tried and found useless and hence have not mentioned. There are many others highly recommended as curative in all cases. These I have not tried, for their alleged unvarying success leads me to the conclusion that they have not been used in the treatment of genuine diphtheria.

It is probable that in a long series of cases, covering several years or several epidemics, no medication now known gives results much better than 85 per cent. of recoveries.